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CHAT WITH OUR CO-SUPPORTERS.

THIS Number of the "British Journal of Anæsthesia" begins Volume Three, and it seems timely to utilize the occasion for a sort of heart-to-heart talk with our Subscribers, for it is to them that most of the success of our venture is attributable, and, by the same token, it is realized that our future lies in their keeping. So it should not be a waste of valuable space to discuss, as it were, certain things *en famille*, especially since the time has arrived to think of taking a step in the direction of increased usefulness.

It is in the nature of things that nothing remains stationary. Things either advance or recede. Likewise, every successful movement has behind it a *vis a tergo* that keeps it going in the proper direction, for, although a good Idea in itself may possess considerable innate strength, there must be a driving force behind it to make it thrive and flourish. This is especially true of a medical periodical for the success of which both the Idea and the Driving Force are equally necessary. The Idea must be a good one, that is, with the field already overcrowded only a Journal which has a distinct *raison d'etre* can find and hold a proper niche for itself. The "British Journal of Anæsthesia" was founded on the Idea that there was a distinct need for a special periodical of its character, and the necessary Driving Force to keep it going has been the large number of anæsthetists willing to give it active, steady and consistent support.

What is the Ideal Journal? There are, no doubt, divergent views to be considered, but as regards ourselves the main desideratum has been that it should prove a true and

helpful record of contemporaneous thought in the field of anæsthesia. It should publish, as *early as possible*, the results of the investigations of workers throughout the world, and, what is of the highest importance, every article should bring the subject of which it treats right up to date. This latter is highly necessary, because text-books, generally speaking, appear tardily, and, in order to keep *au courant*, it is necessary to augment the information they contain by the additions of new facts as they become available. A journal should not merely limit its usefulness to the first reading. *Literæ Scriptæ Manent.* The written word remains, therefore a periodical must be so constituted that it may be placed alongside the text-book on the shelf, to be consulted as occasion requires.

It may be argued that, with somewhat limited space, certain rather standardized features are better left out occasionally. After all, it is not simply "good reading" that is principally required. One can get that elsewhere. True, even much that is written in "heavy vein" is often fulsome, useless and unsubstantial, but it is recognised that an author must have elbow-room to develop the one or more good ideas that his article may contain. We do not begrudge him his extra pages, since we realize that a certain latitude is required even by the most earnest of writers.

And this brings us back to the proposition enunciated in the first part of this little talk—*viz.*, that nothing remains stationary. The "British Journal of Anæsthesia," in passing into its Third Year, is considering a move in the direction of advance. We believe that our matter should see the light of day as quickly as may be, and our aim is to make this possible by *ultimately* appearing *every month*. That is an Ideal; but, alas! it is not practical at present. Nevertheless, it is a star worth hitching our wagon to, and, in the meantime, it is desirable to attempt a compromise—that is, to print an issue *every other month*. We believe that this will soon be feasible, and ways and means to this end are being considered. When the time appears opportune we shall let all and sundry

know. It is sufficient for the present to hold this out as a near possibility—nay, let us say, a probability.

It was once argued that our great difficulty would be in obtaining sufficient matter to keep the Journal going. Time has proved otherwise. Workers in many parts of the world are now, we are pleased to say, thinking in terms of our Journal, and whether it is in London, Edinburgh, Vienna, Königsberg, Paris or New York, the "British Journal of Anæsthesia" is regarded as an ideal medium for broadcasting anæsthetic papers. The stage has thus been reached where it is quite possible to supply our readers regularly with articles by the greatest authorities, and it could probably be done every other month as easily as each quarter, if the necessary additional support can be assured. Let us illustrate a certain phase in the situation. Among the papers we have in our possession is one on Helium Gas. This gas is now being used in airships, its principal advantage being its non-inflammability. The paper is by two of the greatest authorities, and deals with its effects on human beings and mice. This opens up an entirely new field, and its possible connection with anæsthesia is obvious. Our space does not permit of its inclusion in this Number, and by the time our next Number is in print it will already have been published elsewhere. We must content ourselves with merely printing the briefest abstract in this issue. Another paper deals with Endocrinology and Anæsthesia. It is only necessary to mention the fact that the author discusses such a vital subject as the Status Lymphaticus in its anæsthetic relationship. These are not related "to point a moral or adorn a tale," but to simply illustrate a state of affairs which actually exists.

From the first inception of the "British Journal of Anæsthesia" it has been the desire of the founders to emphasize the fact that the entire venture was to be one of mutual undertaking—that is, a something that was to be conducted solely in the interests of the science and art of anæsthesia, while forming at the same time a sort of literary partnership amongst those of us whose work lies along these

lines. It was obvious that if we were to place this branch of Surgery *upon the status which it deserved*, it was necessary for all of us, each in his own measure, to aid where and how possible.

The question may be asked, then, how can supporters of our venture further help in advancing the Journal's interests? There are two sources of support for a periodical. First and foremost are the Subscribers, and these are both private and public. By public is meant hospitals, libraries, universities and societies. These in many cases can be influenced by personal endeavour. It may be merely a matter of a little chat with a librarian. There are still some anæsthetists who are not on our list, a portion of whom would no doubt join us if the case were put to them properly by one who is already a Subscriber.

Secondly, there are the Advertisers. This is a very important section, and, in our case, is practically limited to dealers in purely anæsthetic articles. We have been fortunate in being able to get the support of every leading house on both sides of the Atlantic. We hope this confidence in our ability to help their sales has not been misplaced. Advertisers are business men, and, rightly, expect proper returns for their outlay. It is well for readers to remember this when they are purchasing supplies. The revenue received from this source is valuable, and as they are helping us it is only right to return the compliment when possible.

To conclude, these random notes are written solely with the object of taking our co-supporters into our confidence. As has already been stated, and might be repeated, our ambition is to publish ultimately a monthly Number. The future alone will show if this can be attained. In the meantime, it is hoped that the transition may be made easier by publishing a Number every other month. This, while not quite so ambitious, lies nearer of attainment, and only awaits favourable circumstances to be put into execution. This is our immediate aim, and we hope that we can rely upon every anæsthetist to do what lies in his power to bring about this needful and progressive step.

THE EDITOR.

SACRAL ANÆSTHESIA.

MINOR POINTS OF MAJOR CLINICAL IMPORTANCE.

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SACRAL anæsthesia, as a division of regional anæsthesia, has gained considerable ground in many fields of surgical practice. The progressive surgeon has realized that it is a most valuable aid in the achievement of end-results.

The advantages of local and regional anæsthesia over general narcosis, based on laboratory findings and clinical observations, have been so well demonstrated by Crile, Matas, Pauchet, Braun, Finsterer, Farr, and a host of others, that to review them here would be crowding the literature uselessly. Our aim is, therefore, more usefully directed to such improvements of technique as would render the application of regional anæsthesia easy, sure, and safe in the hands of every surgeon and anæsthetist, thereby helping in its popularity and universal acknowledgment.

The recognition which regional anæsthesia, as a whole, is steadily winning, is evidenced by the recent contributions to the medical literature; the special time devoted to the subject at medical meetings; the particular interest aroused at clinical demonstrations; the desire expressed by many surgeons to familiarize themselves with the method; the anxiety shown by the general anæsthetists to broaden the scope of their activities; and the conspicuous place allotted to the regional method in the department of anæsthesia.

Local anæsthesia, or tissue infiltration, a method whose basic principles imply injection of the operative field as the operation progresses, is being gradually replaced by regional anæsthesia, or nerve blocking, which is a procedure entirely independent of the surgical act. The surgeon is thus enabled to focus all his attention to the operative manipulation and

lose no time in repeatedly passing from the knife to the syringe and *vice versa*. This single technical improvement has been a great inducement to the use of regional anæsthesia in major surgery. The acquired fact that patients can be completely anæsthetized before the operation is begun is largely responsible for the fair trial which regional anæsthesia has been given in recent years. With increased experience, its practical value is being rapidly acknowledged, and the method is winning a well-deserved reputation.

Regional anæsthesia is actually considered one of the most important factors in the solution of the problems presented by the bad-risk patients, especially the aged. Its use in selected cases has brought out the innocuousness of the method and broadened the scope of operability. Its adoption as a routine procedure has minimized morbidity and reduced mortality to its lowest term. Regional anæsthesia represents one more refinement of surgical technique which reduces or eliminates the possibility of complications which are not infrequent following inhalation narcosis, however skilfully administered.

Regional anæsthesia has almost completely superseded general narcosis in certain branches of surgery. In others, its operative usefulness and comparative innocuousness have urged the use of regional anæsthesia where general narcosis was said to be contra-indicated. But the field of contra-indications to inhalation anæsthesia is being so much enlarged that we are justified in expecting in the near future a reversal of the present methods, whereby regional anæsthesia will be used as a routine procedure and general narcosis in exceptional cases.

An excellent illustration of the tendency towards the routine of the regional method is seen in the department of Urology, as evidenced by the Collected Papers of the Mayo Clinic published in the last four years; the extensive space allotted to sacral anæsthesia at the stated meeting of the Medical Association of the Greater City of New York, held on January 21, 1924; the lengthy discussion on local, regional and spinal anæsthesia which took place at the annual meeting of the American Urological Association held at Atlantic City, June, 1924, etc., etc. Most surgeons are now absolutely

certain of the real benefits of regional anæsthesia in Urology. Paravertebral block is being employed with increased satisfaction for operations on the kidney and ureter; sacral block is considered the method of choice for those on the bladder and prostate.

Other illustrations of this tendency towards the routine use of regional anæsthesia are seen in the fields of Proctology and Gynæcology. In fact, Sacral Anæsthesia is conquering everything which comes within its reach.

General Principles.

The general principles of the method all have at their root the scrupulous attention to minor details which in reality are major factors for success.

With all methods of inducing surgical anæsthesia whereby maintenance of consciousness is one of their basic principles, it is essential that the patient be relieved of the psychic strain due to apprehension, fear or anxiety by the use of some form of anociation, as advocated by Crile. The combination of morphine and scopolamine in weak doses has been found to meet this purpose very satisfactorily; and after many years' experience with all types of patients, these doses have been fixed at Morphine Sulphate gr. 1/6 and Scopolamine Hydrobromide gr. 1/300, given hypodermically one hour before the time scheduled for operation. At times it is desirable to repeat this dose in the course of the anæsthetic procedure if the patient's behaviour is not deemed favourable. By all means, however, the patient must be kept under sufficient control. *Narcotics must be used to blunt consciousness, but not to abolish it. Doses should, therefore, be accurate.*

In the hospitals where these solutions are prepared by dissolving tablets in distilled water, it is the author's experience that errors have several times resulted in undesirable symptoms of scopolamine poisoning, among which was delirium. The delirious condition of these patients two or three hours after the operation led to an inquiry about the dose of scopolamine injected and the manner in which the solution had been prepared. In one case two tablets of gr. 1/150 had been used to equal gr. 1/300; in another case three tablets of gr. 1/200 had been dissolved and half the

solution injected; in other cases the nurses were unable to convert accurately 1/200 to 1/300. It is not sufficient to leave instructions to the nurse; but every effort should be made to insure the patient's safety by using these narcotic drugs in such form as can be dispensed without imposing upon the nurse's knowledge of reducing of fractions. The sealed ampule containing one accurate dose of morphine and scopolamine is, we submit, the only rational form.

Anæsthetic solutions are worthy of considerable attention, since half of the success depends upon the accuracy of their concentration. They should be prepared just before use by dissolving accurate doses of the anæsthetic drug in accurate volumes of normal physiologic or saline solution. The conversion of concentrated solutions into weaker solutions is not always reliable.

The *quantity of solution* which it is safe to inject depends not only upon the concentration of the solution, but varies with the type of patient. Doses administered to strong, robust patients may cause undesirable reactions when applied to those of lowered resistance; they should accordingly be reduced to a minimum that is compatible with satisfactory anæsthesia. Procedures requiring the injection of weak solutions should always be preferred when dealing with bad-risk patients. It is obvious, therefore, that many of the doses recommended are subject to variations; but they should not be exceeded with impunity in patients of average surgical resistance.

The *rate of injection* should be carefully controlled, since on it depends the rate of absorption and, consequently, the nature of the patient's reaction to the drug. *The first syringe should be discharged very slowly*, so that time may be allowed for the capillaries to close or the organism to adjust itself before larger doses are injected. Injections may then be made with progressively more speed, but *in no case should the procedure be rushed through.*

The *anæsthetic drug* used is also a noteworthy factor of success which should not be underestimated. Novocaine is unquestionably the drug of choice. Among the various brands of Novocaine is *Neocaine*, a French product of the Corbiere Laboratories, Paris, which the author strongly

recommends, having used it with gratifying results in thousands of cases since 1916. It is put up in capsules of 0.50 grams, which renders the extemporaneous preparation of solutions very easy and controllable. Accuracy in concentration is reached only by using glass measures graduated in cubic centimetres. *Neocaine pure contains no adrenaline.* The addition of adrenaline is thus left entirely to the discretion of the surgeon.

Instruments are factors in the achievement of technical skill. Good technique demands that the sacral canal should never be punctured except with an unbreakable needle. Puncture of the posterior sacral foramina is easier with flexible steel needles. Accuracy of injection and effectiveness of aspiration can be secured only by making air-tight connections between needle and syringe, piston and barrel. The author's outfit has been very carefully designed to meet these specific requirements. The aspiration test for blood or cerebrospinal fluid is effectual only after making sure that the lumen of the needle is patent. It is, therefore, necessary to *inject a few drops of solution before aspirating.*

The *patient's position during the injection is* commanded by the injection technique; the *ventral, with the hips raised,* being the most favourable. The sitting erect position (Läwen) for caudal block is not infrequently a contributive cause of faintness. The lateral position (Cathelin) does not lend itself very easily to caudal block and gives a distorted picture of the superficial landmarks for Transsacral block.

The patient's position after the induction of anæsthesia is subordinate to so many factors, such as age, blood-pressure, condition of the heart and lungs, type of operation, local operative conditions, etc., that no hard and fast rule can here be given. Individual experience is the only guide. It is advisable, however, that the *horizontal position, or better, a slight Trendelenburg* be maintained during two or three hours following the operation.

In painting the skin with tincture of iodine, alcohol, ether, alcoholic solution of picric acid, etc., care should be exercised that no excess of solution spills upon the genitals or anal region. With such carelessness the patient may experience much discomfort during the accomplishment of the anæsthetic

procedure, until these regions have been rendered insensitive.

Gentleness of the operating team, the use of soft cushions and pads on the operating table, and avoidance of unnecessary noise with the instruments are also factors of success. Leaning on the patient's chest, clinging to his limbs, or keeping an instrument tray on his abdomen during perineal operations are the acts of poor technicians.

Definition and Terminology.

Sacral anæsthesia is generally understood to mean the state of analgesia following the injection of the sacral nerves. This can be accomplished by three well-known procedures which, although intended for identical purposes, differ widely in their characteristic features, that is anatomically and technically, and have respective indications and limitations. These procedures are known as (1) Caudal or Extradural block, (2) Presacral block, and (3) Transsacral block.

(1) *Caudal or Extradural* block consists in depositing the solution at the caudal extremity of the spinal column, within the sacral canal, outside the dura mater. It is thus distinguished from intradural or intraspinal block which results in what is commonly called spinal anæsthesia. The term "epidural anæsthesia" is still used to express the idea of extradural injections; but it has long been disapproved by Cathelin, the originator of epidural injections by puncture of the sacral canal.

(2) *Presacral block* implies injection on the anterior aspect of the sacrum. It is called "parasacral" by Braun, to whom the method is accredited.

(3) *Transsacral block* is accomplished by carrying the injections through the posterior sacral foramina to the anterior primary divisions of the sacral nerves. It is sometimes called "parasacral."

The use of the term "parasacral" to denote indifferently Transsacral or Presacral block leads to undesirable confusion, which can only be avoided by using the words "transsacral" or "presacral" in their proper places. The procedures of Caudal, Transsacral and Presacral block have been so well defined that there seems to be no reason why such expressive

terminology should not be respected, especially for didactic purposes.

Caudal Block.

A few important changes have been brought into the technique originally described by Cathelin for the puncture of the sacral canal and that advocated by Läwen for producing surgical anæsthesia. The best position is that in which the patient lies flat on his stomach with a cushion under the hips. The pelvis is thus raised and the landmarks rendered more accessible. The needle used is a malleable nickeloid needle which bends but will not break within the sacral canal. It is the same as that used and recommended by the author for intradural block. The best solution is a 2 per cent. neocaine solution when caudal block is the only procedure used to anæsthetize the sacral nerves. The addition of potassium sulphate, sodium bicarbonate, etc., etc., to the anæsthetic solution does not markedly improve the quality of the anæsthesia. *Adrenalin* is the only substance that is routinely added to the Neocain solution. For sacral anæsthesia it is customary to add not more than ten drops (about seven minims) of adrenalin solution (1 : 1000) to 100 cc. of the anæsthetic solution. A colourless solution of adrenalin is the only one that should be used.

The simplicity of technique for puncturing the sacral canal was the primary cause of the popularity of sacral anæsthesia; but the percentage of failures noted with caudal block, although varying with individual experience, is still high. These failures are chiefly due to the following causes:—

1. Inability to enter the sacral canal through lack of knowledge of the anatomical variations of the sacrococcygeal region;
2. Impossibility to enter the canal through absence of accurate landmarks;
3. Decreased permeability of the neural sheaths of the sacral nerves;
4. Injection of too weak solutions;
5. Increased capacity of the sacral canal, due to the size of the bone;
6. Defective position of the needle in the canal;

7. Bleeding within the canal, as the result of accidental trauma inflicted by the needle during its penetration;
8. Defective closure of the sacral canal rendering its penetration and injection dangerous;
9. Injection on the posterior aspect of the sacrum;
10. Impermeability of the sacral hiatus.

Greater than has been published in text-books of anatomy is the number of cases in which fusion of the first coccygeal vertebra with the sacral bone changes the aspect of the sacral hiatus and converts it into a "sacrococcygeal hiatus." In such cases there are four cornua instead of two, the lower ones belonging to the coccyx and being very often the more prominent. The same picture is present when the laminæ of the two last sacral vertebræ have not closed and the sacrum not fused with the coccyx. In both cases the vertex of the hiatus is more or less occupied by a thin bony flap which obliterates the normally deeper and more penetrable part of the hiatus; thus compelling puncture through a site lower than the middle point of the classical triangle. With a slight turn of the hand, the canal can be entered with relative ease.

Absence of sacral cornua is exceptional. Palpation of the sacrococcygeal region occasionally fails to give accurate information, because the cornua are comparatively flat or the thickness of the soft structures overlying them abnormally great. When the hiatus cannot be defined with sufficient precision by palpation, it can always be located by exploration with a needle. A thin steel needle 50 mm. long with very sharp point, such as that contained in the author's set, is best suited for this exploration, because it is less traumatizing than the regular nickeloid caudal block needle and has the advantage of interfering very little with tactile sense. The following is the exploration technique:—

An intradermal wheal is raised at the extremity of the gluteal cleft, on the median line and over the region where the sacral hiatus would normally be situated. The 50 mm. steel needle, previously attached to the syringe filled with solution, is passed through the wheal and repeated punctures are made fanwise, exactly in the median plane of that region, searching with the point of the needle for the special fibrous

resistance of the sacrococcygeal membrane. As soon as its presence is suspected, moderate pressure is applied along the axis of the needle, which then pierces the membrane and comes in contact with the anterior wall of the sacral canal. The notion conveyed to the fingers is that of membranous resistance immediately followed by free space, then bony contact. A few drops of the anæsthetic solution are injected each time that the needle comes in contact with the harder structures in the depth. When the exploring needle has thus located the hiatus, its direction is noted and the nickeloid unbreakable needle substituted for the steel one, in order to proceed with the usual caudal block technique.

Impermeability of the sacral hiatus is exceptional. It is a condition resulting from excessive flattening of the posterior wall of the canal which brings both walls almost in contact with each other. The presence of a spur on the anterior wall of the canal (defective fusion) may also prevent the needle from passing. Calcification of the sacrococcygeal membrane is, we believe, merely theoretical. We have seen it neither at the amphitheatre nor in the operating room.

The sacral canal should always be punctured exactly in the middle line. Slight deviation of the needle may cause uneven distribution of the anæsthetic solution with consequent unilateral anæsthesia; and it is especially in large sacral bones that such a defective injection is likely to be made.

Bleeding within the sacral canal may follow a single penetration of the needle. It may be due to the rupture of one of the blood-vessels in the central portion of the canal; but it is more frequently the result of scratching the periosteum of the posterior wall of the canal. *It is unsafe to inject while the canal is bleeding.*

The presence of an *unusually large hiatus,* as the result of the non-closure of the lower half or two-thirds of the posterior wall of the sacral canal, should not be overlooked. In such cases *caudal injection should be made with great caution and occasionally avoided,* because it may prove dangerous to life, owing to the risk of puncturing the dural sac and making an intradural injection.

The lower extremity of the dural canal, otherwise called

dural sac or *dural cone,* which encloses the subarachnoid space with the cerebrospinal fluid contained in it, *ordinarily stops between the second and third sacral vertebræ.* This level can be traced on the surface of the skin by a *straight line joining the posterior superior iliac spines together.* The depth of the puncture of the sacral canal is thus controlled with great accuracy. *If the site of puncture, conditioned by the anatomical features of the hiatus, is in too close proximity to that line, it is safer to abstain* from puncturing the sacral canal. It is then that Transsacral block assumes the highest practical value.

The aspiration test for cerebrospinal fluid may be negative, yet the needle may have punctured the dural sac. This is probably due to the partial collapse of the dural cone, as the cerebrospinal fluid gravitates to the more dependent portion of the dural canal. If the needle follows the posterior wall of the sacral canal, there will be very little risk, if any, of puncturing the dural cone; but the safest technique is to *determine the distance between the sight of puncture and the extremity of the dural cone, and gauge the depth of puncture in terms of that distance.*

From the foregoing discussion of the causes of failure to induce sacral anæsthesia by puncture of the sacral canal it can be realized that the percentage of failures may be considerably reduced with a thorough knowledge of anatomy and the scrupulous attention to minute details of technique. It is also obvious that there are failures which are unavoidable and uncontrollable, to which no remedy will ever be brought.

The delayed anæsthesia associated with caudal block would be but a slight drawback, were it not for the fact that it is only after waiting at least thirty minutes that a failure can be pronounced. To obviate the inconvenience caused by failures of any origin, a much better procedure is to perform the transsacral block during the waiting period of the caudal block. The association of both procedures, which the author calls "sacral block," never fails to produce the desired result. It takes only from 15 to 20 minutes to be accomplished, and the operation may be begun immediately after the last injection.

Caudal block is a safe procedure when it is used with the intention of inducing sacral anæsthesia. It is not free from

danger when attempts are made to reach higher regions. The question of high epidural injections by puncture of the sacral canal is of too great importance to be discussed as one of the minor points brought out in this paper; but we believe that it is our duty to sound a note of warning against the accomplishment of such a procedure, at least at the present stage of development of technique.

Presacral Block.

Injection of the anterior aspect of the sacrum at the level of the sacral foramina is associated with the risk of puncturing the rectum, spreading infection and transplanting cancer cells.

Variation in the angulation and curvature of the sacral bone make the injection of the first sacral nerve occasionally very hazardous. The posterior primary divisions of the sacral nerves are not reached with this procedure, unless the needle is passed through each foramen, which is a very painful manipulation. There are, besides, no accurate landmarks for reaching the foramina. We have used presacral block a number of times in plastic operations on the vagina, transperineal prostatectomy and hæmorrhoidectomy; but we are not prepared to recommend its routine use. It is, however, desirable that the technique of presacral block be mastered, so that the procedure may be used if pathological conditions of the posterior sacral region should render transsacral block impracticable and other serious considerations make spinal anæsthesia inadvisable. In any case, presacral block should be accomplished with great caution, dexterity and skill.

Transsacral Block.

Transsacral block comes to the rescue when caudal block cannot be accomplished or fails to produce the desired result. It is a procedure accredited to Danis, of Belgium, who devised it in 1913. Since then, it has been used extensively in France by Pauchet, Sourdat, de Butler d'Ormond and the author, who developed the technique to a high degree of precision and demonstrated its practical value as a routine method of anæsthesia. With closer familiarity transsacral block is winning greater popularity.

The classical landmarks for blocking of the sacral nerves by the transsacral route are the posterior superior iliac spines and the sacral cornua. *The posterior superior* iliac spine referred to in connection with transsacral block, *is not the anatomic spine*, which in the majority of cases cannot be defined by palpation; *it is the most prominent point of the posterior extremity of the iliac crest, and is about one centimeter higher than the anatomic spine.* The second sacral foramen lies one centimeter medial to and below that spine which we propose to call the "regional posterior iliac spine." Its position is constant and may be traced with great accuracy on the surface of the skin, except in exceedingly fat patients.

The so-called sacral cornua do not always belong to the sacrum, as explained in the first part of this paper under CAUDAL BLOCK. They are not always two in number. There may be two, four, six, more or less prominent on the sides of the hiatus, according as the first coccygeal vertebra has fused with the sacrum or the laminæ of the last two sacral vertebræ have failed to unite on the median line. If the position of the hiatus and its vertical dimension are defined with accuracy, the level of the lowest cornua in relation to the gluteal cleft is instructive. When low, it indicates addition of the first coccygeal vertebra to the sacrum with five sacral foramina; when high, it marks non-fusion of the laminæ. Even here the coccygeal cornua may be so prominent as to be misleading. Both conditions may, however, be present and associated with five sacral foramina; in which case the lowest cornua are coccygeal and not sacral. Our personal examination of cadavers and observation of patients have demonstrated the fact that in the male the four cornua type of hiatus is more frequent than the classical two cornua type. *In all descriptions, the meaning of sacral cornua should be taken as the lowest lateral tubercles, one on each side of the sacral hiatus, in the sacrococcygeal region.* The fifth sacral foramina are thus situated laterally to and a little above these tubercles. Too great stress cannot be laid on the accuracy with which the superficial landmarks should be taken, since on them entirely depends the location of all the sacral foramina. In cases of only four sacral foramina the third is obviously injected twice most of the time.

It is of paramount importance to bear in mind that in the adult the thickness of the sacrum decreases from above downwards in the proportion of 3 cm. to 0.5 cm. at the levels of the first and last sacral foramina respectively, and that the posterior and corresponding anterior sacral foramina face each other and form the orifices of a canal in which the solution is deposited. It is, therefore, easy for the needle to pass into the pelvis and reach the organs contained in it. A distended bladder may be punctured by passing the needle deeply enough through the second posterior sacral foramen. The rectum may be reached more easily through the last two pairs of foramina. Sight should not be lost of the aforementioned anatomical features of the sacrum, and care *should be exercised not to allow the point of the needle to pass the anterior aspect of the bone.*

Puncture of the second foramen is the easiest on account of its constant position. It should be the first attempted. The aspiration test should always be made before injecting, since the puncture of a blood-vessel through a sacral foramen, especially the first and second, is not infrequent. If there is bleeding, immediate injection is objectionable; but puncture of that foramen may be repeated, and if at this time the aspiration test for blood is negative, injection can be made with safety. Access to the first sacral foramen, which is situated 2.5 cm. to 3 cm. above the second, is occasionally rendered difficult by the presence of a bony flap obliterating most of its medial portion. The needle must be passed laterally to the flap, and not infrequently over its upper margin. If the needle fails to enter the first foramen after repeated gentle and accurate punctures, a second injection in the second foramen ordinarily reaches the desired aim. Scrupulous attention to these apparently minor details are of major importance in safeguarding against pitfalls and insuring success.

Sacral Block.

Sacral block is the name given by the author to the association of Caudal block and Transsacral block (Labat. "Regional Anæsthesia: Its technique and clinical application." W. B. Saunders Co., Philadelphia and London, 1922.)

Its technique consists in injecting the sacral canal with from 20 c.c. to 30 c.c. of a 1 per cent. *Neocaine* solution, immediately followed by the injection of the sacral nerves individually through the posterior sacral foramina with varying quantities of the same 1 per cent. solution. The results are remarkable in that the anæsthesia is perfect soon after the last injection has been made. It takes only from 15 to 20 minutes to accomplish the whole procedure. Failures to obtain anæsthesia are exceptional. They are registered only in such operations as require tugging on the broad ligaments or rough manipulation of tissues which are related directly or indirectly to the lumbar nerves passing through the pelvis or to those originating from the hypogastric plexus. Such results cannot be ascribed to defective anæsthesia of the operative field.

Sacral block is the ideal method for all operations on the anus, perineum, vagina, cervix uteri, urethra, prostate, bladder and rectum. For resection of the rectosigmoid it must be completed by paravertebral block of the last three lumbar nerves, as already demonstrated by the author at the Mayo Clinic. (*Labat. Posterior resection of the rectum and rectosigmoid. Johns Hopkins Hospital Bulletin, April,* 1922.) All plastic operations on the vagina and cervix uteri are painlessly performed with sacral block; but vaginal hysterectomy and the Mayo-Watkins interposition require a wider field of anæsthesia similar to that used for resection of the rectosigmoid.

After-Effects.

The untoward effects associated with Sacral Block are negligible when the correct technique is used. The pulse-rate is more or less increased during the early part of the injection within the sacral canal; but if the injection is made very slowly, conditions return to normal within the next two or three minutes. Pallor of the face is so frequent that it may be said to be the rule. Both symptoms may be attributed to the action of the adrenalin contained in the neocaine solution or to that of the anæsthetic drug discharged in the general circulation just before the action of the adrenalin on the capillaries. We believe that they are normal reactions

constituting the natural defense of the organism, because of their presence in almost all cases. *They are not alarming and do not justify the use of stimulants. We only advise slight Trendelenburg position and a large cup of hot black coffee, taken by sips, as soon as the patient is brought back to his bed after the operation.*

Nausea is rare; vomiting exceptional. If nausea is present, the only way to prevent vomiting is to close the patient's nose and make him breathe deeply through the mouth, in and out, regularly. Prevention of vomiting keeps the patient in fine shape and the surgeon in excellent mood.

Sloughing of the tissues overlying the sacrum is due to the injection of large quantities of anæsthetic solution on the posterior aspect of the sacrum. Infection is chiefly due to accidental puncture of the rectum. Such mishaps are the result of very poor technique.

Discussion.

With the introduction of regional anæsthesia into surgical practice, many of the drawbacks of local infiltration have disappeared, especially those of loss of time and risk of infection. Expert clinical demonstrations given in recent years have largely contributed to win confidence in the regional method and widespread its use.

Successful induction of sacral anæsthesia depends upon many factors, among which are:—

(1) Thorough knowledge of the anatomic variations of the sacrococcygeal region;
(2) Strict observance of the basic principles of the method;
(3) Scrupulous attention to details of technique;
(4) Thorough understanding of the limitations of caudal block; and
(5) Skilful accomplishment of the sacral block procedure.

Like all other surgical procedures, the successful application of sacral block depends greatly upon individual skill in using it. It has, however, been studied from so many angles and its technique simplified to such a degree that it can now be used routinely with safety and ease.

To be able to decide upon the merits of the method needs considerable experience which cannot be acquired by brief study, superficial knowledge or hearsay. The basic principles of regional anæsthesia are so different from those of inhalation narcosis that to establish fair comparisons between the two methods requires expertness in both. Such comparisons are, however, of common occurrence and almost unavoidable, inherent as they are to past surgical training.

Operating surgeons are the sole judges of regional anæsthesia, as they have been of the other methods in the past. In fair justice to the method, surgeons should familiarize themselves with its fundamental principles, even though they have no intention of injecting their own cases, because they must see that the evidence adduced is the expression of truth and not a misrepresentation or distortion of actual facts mostly as the result of ignorance. They should also encourage the younger men to acquire experience along that line by affording them abundant clinical material, for they also will be the judges of the next decade. On the surgeons' decision depend the future of the method and the welfare of generations of patients.

The practical value of regional anæsthesia is considered chiefly and primarily in the light of the results obtained in the operating room. The operative facilities afforded, as compared with inhalation narcosis, are the outstanding factors for its recognition or dismissal, and it is only when the method has stood the test of the operation that its post-operative merits become worthy of consideration.

With this attitude of its judges—an attitude which we believe is quite legitimate—regional anæsthesia should be studied thoroughly before any attempt is made to give clinical demonstrations of its practical value. Even then, leniency should be asked on account of its youth.

The place of honour to which regional anæsthesia is ascending is the result of accumulated evidence that the method has greater merits than inhalation narcosis. Sacral anæsthesia will reach the goal when it is universally realized that the strict observance of minor details of technique is of major clinical importance in the achievement of operative success.

ANÆSTHESIA IN DIABETES.*

By I. M. Rabinowitch, M.D., C.M.

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I WISH to discuss the influence of general anæsthesia on the metabolism of the diabetic individual. It is hardly necessary to consider, in a statistical manner, the influence of improved methods of treatment on the diabetic as an anæsthetic risk. The data which have accumulated at the Montreal General Hospital would probably differ very little from those of any other well-equipped hospital. Death due to diabetic coma, brought on by anæsthesia, is, and should be a rare occurrence. It should, however, be emphasized that, in spite of all our improvements, general anæsthesia is still to be avoided if at all possible, and where not possible, the duration of the anæsthesia should be reduced to the minimum. The whole story of diabetes is not yet told. Since the advent of insulin, coma may be more satisfactorily treated. Treatment should, however, as in the days prior to insulin, aim at preventing rather than combating this condition.

Of the group of substances which paralyze sensation, but do not abolish consciousness—namely, novocaine, cocaine, etc.—little need be said. These are found to be comparatively safe, when attention is paid to the avoidance of undue trauma and infection. The chief problem with which we are now concerned is the use of chloroform, ether and nitrous oxide. A brief consideration of the influence of these drugs on the metabolism in the normal subject is necessary in order to appreciate the possible disturbance which may occur in the diabetic.

* Delivered before the Congress of Anæsthetists and International Research Society, Atlantic City, May 27th, 1925.

In the normal individual these substances may produce an alteration in the metabolism of both carbohydrates and fats. This is manifested by hyperglycæmia, and the presence of products of incomplete oxidation of fats (acetone, diacetic acid, etc.) in the urine. Chloroform seems to have a specific influence on the oxidation of these products. Joslin points out Blum's observation of this phenomenon in animals. When beta oxybutyric acid was injected into a dog, it was completely oxidised. When, however, the same experiment was repeated while the animal was under the influence of chloroform, incomplete oxidation of the acid was noted.

Ether may not only prevent the conversion of glucose into glycogen, and storage of the latter in the liver, but may also interfere with the metabolism of the glycogen already present there. Hyperglycæmia results. Ether may also produce an acidosis. Stehle and Bourne, and others, have shown that in animals the administration of ether may increase the hydrogen ion concentration of the blood, and lower its CO_2 combining power. Corresponding results are noted in man.

Ether may also impair the efficiency of the kidneys, even to the extent of causing suppression of urine, partial or complete. Under such a condition, an acidosis, if present, would become more marked. To the acidosis produced by the formation of new acids (due to diabetes), would be added one due to the retention of normally formed acids.

Another manifestation, and probably of equal importance, and due to kidney damage, may be defective ammonia metabolism. Observations reported by Nash and Benedict on animals, and by me on man, strongly suggest that one of the functions of the kidney is to form ammonia. If ammonia formation is one of the functions of the kidney, the possible dangers of a disturbance of this function caused by anæsthesia, appear obvious, when one considers the part that ammonia may play in the regulation of the reaction of the body fluids. At pH 4.8, a urine reaction, one molecule of phosphoric acid is combined with less than one molecule of base as acid sodium phosphate. At pH 7.45—the reaction of the blood, one molecule of phosphoric acid is combined with about 1.8 molecules of base. Thus in the excretion of

one molecule of phosphoric acid, nearly one molecule of base may be retained within the body. The production of one molecule of ammonia represents the saving of one molecule of base. Therefore, in the presence of a kidney lesion associated with decreased ammonia formation, the acids formed have to call upon the other reserve bases of the body. Under such conditions an acidosis may be more readily produced. Defective ammonia production accounts to some extent for the fact that, at times, the urinary findings following ether anæsthesia, may be identical with those found in nephritis.

With these few observations of the effects of anæsthesia on the metabolism of normal individuals, we can more readily appreciate its possible profound effects on the diabetic. The fundamental disturbance in the metabolism of the diabetic is a departure from the physiological condition involving the capacity of the organism to metabolize carbohydrates. This departure may involve either the mechanism of oxidation, the mechanism of storage, or both. Carbohydrates are necessary for the complete oxidation of fats. These carbohydrates must not only be present, but be in a process of oxidation. In the absence of a sufficient quantity of oxidizing carbohydrate, products of incomplete fat oxidation are produced—namely, acetone, diacetic acid, and beta oxybutyric acid. If present in sufficient quantity these acids produce coma. The ratio of grams of fatty acids to the grams of glucose beyond which incomplete oxidation takes place and acetone bodies are produced is known.

Even in a normal individual the store of glycogen in the body is limited, and will not allow a great degree of starvation before acetone and diacetic acid may be detected in the urine. In a diabetic requiring surgery, this limited store of glycogen may be found greatly reduced, due either to (*a*) severity of the diabetes, (*b*) pre-operative restriction in diet, (*c*) post-operative restriction in diet, and (*d*) inability to take food, due to nausea and vomiting, resulting from either the operation or the anæsthesia. The ease with which an acidosis may result is thus obvious.

These few observations suggest the underlying principles by which the treatment should be guided. These consist of (*a*) obtaining a state of metabolism approximating, as closely

as possible, the normal, before administration of the anæsthetic, (*b*) choosing that anæsthetic which, for the period necessary, will produce the least harmful effect, (*c*) supplying a sufficient carbohydrate store for the unavoidable starvation period following the anæsthetic, and (*d*) maintaining that store in a readily oxidizable state.

The three requisites for safe anæsthesia are (*a*) an index of a normal state of metabolism, (*b*) an index of the extent of glycogen storage, and (*c*) a means of promoting the storage of carbohydrates in an individual in whom this function is impaired.

Generally, it may be stated that a normal blood sugar is one of the best indices that the carbohydrate metabolism of the diabetic approximates that of the normal individual, and that the sugar present is in a readily oxidizable state. Hyperglycæmia indicates that there is a disproportion between the rate of absorption of glucose from the alimentary canal or production in the body, and the rate at which it is being oxidized or stored. Hyperglycæmia denotes a disturbance in the metabolism of carbohydrates. Treatment should, therefore, aim at obtaining a normal blood sugar before the anæsthetic is administered.

A normal blood sugar may be an index of a normal state of carbohydrate metabolism at the time of the test, but it is no index of the amount of sugar available for the future. Clinically, this may be judged approximately by the carbohydrate content of the diet, and the metabolic rate of the individual for a few days prior to anæsthesia. The milder the diabetic, the more liberal is the carbohydrate allowance, and therefore the greater the storage. The rate of metabolism of the individual should also be considered. It may be generally stated that, whatever the cause, fever, etc., the greater the metabolism, the less will be the carbohydrate stored.

A liberal carbohydrate store may be obtained either by diet alone, or by diet combined with the use of insulin. The proper procedure to follow will depend upon the severity of the diabetes. In case of emergency administration of glucose combined with insulin may be necessary. In either case hyperglycæmia should be prevented.

If insulin is necessary it should be given at such time before the anæsthetic that its beneficial action may be noted, *before* the harmful effect of the anæsthetic may be produced. Coma is more easily prevented than treated. Diabetic coma following ether, once produced, may be combated only with the greatest difficulty, or not at all, in spite of enormous dosage with insulin. The following cases are cited in illustration :—

Case 1. A mild diabetic. The urine was free of sugar and acetone bodies before operation, and the blood sugar was normal, 0.126 per cent. The operation consisted of a cholecystectomy, necessitating two hours' ether anæsthesia. The urine the following day showed sugar and acetone bodies, and the blood sugar was 0.204 per cent. The condition was readily controlled with insulin, and the following morning the urine was sugar free and the blood showed only a slight hyperglycæmia (0.154 per cent.). The acetone bodies had disappeared from the urine. Thus :—

		Blood Sugar %.	Urine Sugar.	Urine Acetone Bodies.
Feb. 16	Operation..	0.126	0	0
„ 17	.....	0.204	6.3 gms.	Present
„ 18	.....	0.154	0	0

The ease with which the metabolism of a diabetic may be disturbed is apparent. In this case the hyperglycæmia was readily controlled. The difficulty, however, with which hyperglycæmia may be at times controlled, unless insulin is given early, is shown in the following case :—

Case 2. A mild diabetic. The urine was free of sugar and acetone bodies before the operation, and the blood sugar was only slightly increased (0.139 per cent.). The operation consisted of a repair of abdominal hernia, necessitating two hours' ether anæsthesia. The blood sugar noted after the anæsthetic was 0.188. Insulin was then given. No effect was noted two hours later. Only with repeated doses of insulin had the blood sugar returned within normal limits six hours

after the anæsthetic, in spite of the fact that the diabetes was not regarded as severe prior to the anæsthetic. Thus:—

		Blood Sugar %
9 a.m.	.....	0.139
11 a.m.	Ether	
2 p.m.	.....	0.188
	10 units insulin	
4 p.m.	.....	0.186
	10 units insulin	
6 p.m.	.....	0.166
	10 units insulin	
8 p.m.	.....	0.128

A much better procedure, and one yielding better results, is that noted in the following case:—

Case 3. Diabetic gangrene. 55 mins. ether anæsthesia. Amputation of leg.

		Blood Sugar %
8 a.m.	.....	0.270
	30 units insulin	
9 a.m.	.....	0.178
10 a.m.	.....	0.129
	Ether	
11 a.m.	.....	0.102
12 noon	.....	0.100

In this case it will be noted that the anæsthetic was not commenced until the blood sugar was reduced to the normal level. Hyperglycæmia did not subsequently develop. In isolated instances, however, in spite of the precautions taken, hyperglycæmia may subsequently develop, and be controlled with difficulty.

It will be noted that I have made no mention of the study of the plasma CO_2 combining power, as an index of an approaching acidosis. Though it is not of great assistance otherwise, in the treatment of diabetes, anæsthesia complicates the interpretation of results and limits its value. The finding of a low CO_2 combining power in the plasma does not necessarily always denote acidosis. The lowered CO_2 combining power may be due merely to the increased pulmonary venti-

lation accompanying anæsthesia. In increased pulmonary ventilation an excessive amount of carbonic acid is taken from the plasma and alkali passes into the tissues to maintain the $\frac{H_2CO_3}{NaHCo_3}$ ratio constant. The following cases demonstrate this.

Case 1. Diabetes mellitus. Herniotomy. Ether anæsthesia 35 min.

	pH	CO_2
Before operation	7.48	60.2
90 minutes after	7.42	51.3
180 „ „	7.46	35.6
Following morning	7.40	58.3

In this case it will be noted that there was no material lowering of the pH, but the CO_2 combining power of the plasma was lowered from 60.2 to 35.6 volumes per cent. in three hours. The following morning the value for the CO_2 approximated that prior to the administration of the anæsthetic. Since the patient received no alkali during the interval, it would be difficult to account for the rise noted the following morning, other than by a reappearance of the alkali which was taken up by the tissues the previous day during the period of increased pulmonary ventilation. That the above state of affairs is different from that noted in true acidosis, the following case demonstrates:—

Case 2. Diabetes Mellitus. Cholecystectomy. Ether anæsthesia 2 hrs. 10 mins.

	pH	CO_2
Before operation	7.36	56.2
90 minutes after	7.30	40.4
180 „ „	7.19	32.1
Following morning	7.23	37.6

Here both the pH and the CO_2 values were lowered, during the period of observation. The following morning both the

pH and CO_2 combining power were still below that noted prior to anæsthesia.

There are certain other factors to be considered which may influence the course of a diabetic in spite of all the precautions referred to. Of particular note here are (*a*) age, (*b*) duration of the diabetes, (*c*) infection, and (*d*) the use of drugs commonly employed in surgery.

Generally, children and those who have had diabetes for a short time tolerate anæsthesia better, and have their carbohydrate tolerance less disturbed than elderly individuals, or those who have had the disease for a long period of time. It is interesting to note here that both children, and those who have had the disease for a short time, respond more readily to insulin treatment.

Infection is probably the most difficult problem to deal with. Not only are diabetics with infection properly prepared for anæsthesia with greater difficulty, but their metabolism is more readily disturbed. Once disturbed by the anæsthetic, it may require enormous doses of insulin to control the disease. These individuals require special attention. This applies alike to cases of infection with or without fever, as the following cases show.

In diabetes complicated by infection, the "blood sugar time curves" obtained following the administration of insulin present one characteristic phenomenon—namely, the expected fall in blood sugar appears late, and the rate of decrease is unusually slow. Various types of curves may be noted. In isolated instances with septicæmia and severe acidosis, only the slightest changes may be noted in spite of the enormous doses of insulin (100 units) administered. Thus:—

Hosp. No. 3928. Female aged 50. Infected gangrene of right leg. Septicæmia.

Time.		Blood Sugar %.	
Before administration	.....	0.357	Insulin 100 units.
1 hour after „	.....	0.344	
2 hours after „	.....	0.333	
3 hours after „	.....	0.285	
4 hours after „	.....	0.302	

Fever seems to be an influencing factor in the great majority of cases. In two cases with infected gangrene the "blood sugar time curves" were obtained before and two weeks after amputation of the gangrenous extremity. The following are the data:—

In Case No. 5260-23 the fever persisted in spite of the amputation.

In Case No. 146-24 the temperature had returned to normal. Thus:—

Hosp. No. 4260-23 M. Age 65. Infected gangrene of leg. Before operation Temp. 101.

Time.	Blood Sugar %.	
Before administration	0.285	Insulin 20 units.
1 hour after „	0.250	
2 hours after „	0.251	
3 hours after „	0.252	

Two weeks after operation, Temp. 102.1.

Time.	Blood Sugar %.	
Before administration	0.285	Insulin 20 units.
1 hour after „	0.210	
2 hours after „	0.212	
3 hours after „	0.212	

Hosp. No. 146-24 M. Age 57. Infected gangrene of foot. Before operation, Temp. 102.4.

Time.	Blood Sugar %.	
Before administration	0.385	Insulin 20 units.
1 hour after „	0.332	
2 hours after „	0.278	
3 hours after „	0.277	

Two weeks after operation, Temp. 98.2.

Time.	Blood Sugar %.	
Before administration	0.196	Insulin 20 units.
1 hour after „	Missed	
2 hours after „	0.097	
3 hours after „	0.036	Hyperglycæmia reaction.

It will be noted that in patient No. 146-24 following operation and in the absence of fever, a characteristic curve was obtained.

Though in the greatest number of such cases the sluggish response to insulin is associated with fever, such reaction may be noted in the absence of fever, in the presence of suppuration.

Hosp. No. 5951-23 M. Age 59. Abscess thigh. Profuse suppuration. Temp. 98.4.

Time.	Blood Sugar %.
Before administration	0.526 Insulin 60 units.
1 hour after „	0.500
2 hours after „	0.476
3 hours after „	0.500
4 hours after „	0.501

The routine procedure at the Montreal General Hospital is to determine the blood sugar frequently, not only throughout the period of anæsthesia, but also throughout (hourly if necessary) the remainder of the day of operation. The inconvenience both to the patient and to the laboratory may be well repaid by this procedure.

With reference to the influence of drugs on the course of the diabetic, particular mention may be made here of morphine, pituitrin and adrenalin. Morphine is frequently employed with beneficial effect preliminary to operation in normal individuals. Stehle and his co-workers have shown that morphine not only lessens the tendency of ether to increase the hydrogen ion concentration of the blood, but may actually cause an increase in the alkali reserve. Excluding the beneficial effect for other reasons (psychic, etc.), the value of the use of this drug on account of this action alone is apparent. When used in a diabetic, however, consideration must be given to its possible harmful influence on the metabolism of carbohydrates. Hyperglycæmia following morphine administration is a generally recognised phenomenon.

Adrenalin, so frequently employed in surgery, readily produces hyperglycæmia. Eadie has deduced a mathematical expression by which he measures the antagonistic action

between insulin and adrenalin. Therefore, wherever its use is considered and insulin is also required, this antagonistic action must be borne in mind.

Burn has demonstrated the neutralizing influence of pituitrin upon the hypoglycæmic action of insulin. When pituitrin and insulin are administered simultaneously, not only may no effect of the insulin be obtained, but an actual increase in the blood sugar may be noted.

In this attempt to outline briefly the problem of the use of anæsthesia in the diabetic, I hope I have succeeded in emphasizing the one point I intended to. In spite of the advent of insulin, and improved methods of treatment, careful consideration should be given to all the factors involved before anæsthesia is undertaken, and then the duration should be reduced to a minimum.

A NEW ETHER.

The British Drug Houses, Ltd., have recently brought prominently to the attention of anæsthetists their Æther Puriss, B.D.H., which is said to be absolute ethyl ether, and for which is claimed the advantage of producing anæsthesia without nausea. This ether has for many years been freshly distilled as required, but now it is asserted that it has been found possible to produce an article which is quite stable and may be depended upon at all times. In order to obtain these results it was necessary to eliminate both peroxide and acetaldehyde, and this was brought about by improved methods of purification and storing. An exhaustive paper on the subject of the preservation of anæsthetic ether was read before the British Pharmaceutical Conference, Bath, July 21—24, 1924.

A meeting of the Anæsthetic Section, Royal Society of Medicine, Dr. H. E. G. Boyle in the chair, was held at Manchester on June 27th. Dr. H. P. Fairlie read a paper on Chloroform, which was followed by a discussion; and Dr. S. R. Wilson conducted an anæsthetic demonstration in the Physiological Laboratory of the Manchester University.

The Annual Meeting of the Editorial Board, *British Journal of Anæsthesia,* was held in Manchester on the same date.

SOME RELATIONS OF METABOLISM TO PREMEDICATION AND ANÆSTHESIA*

By E. I. McKesson, M.D., and F. W. Clement, M.B.,
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FOR some years physiologists and internists have been interested in the relation of the metabolism or rate of oxidation in the body under the influence of various diseases. They have been searching for drugs which will best control abnormal metabolic rates. And when abnormal metabolism affects the surgical case we, as anæsthetists, are confronted with the problem in a serious clinical way also. We need to know more about basal metabolism, and the effects of anæsthesia surgery, and drugs, upon metabolism, to properly act as consultants, and to intelligently evaluate our patient's condition during an operation.

For the present discussion the expression "metabolic rate" refers to the rate at which oxygen is consumed, absorbed or combusted by the patient. The test is made by measuring the amount of oxygen absorbed in a period of time, by a suitably calibrated apparatus through which the patient breathes.

Let us enumerate some of the factors which affect metabolism. In health the three great factors which affect metabolism and cause increased O_2 absorption are (1) physical exercise, (2) digestion of food, (3) exposure to cold. Eliminate these factors and we are prepared to obtain the basal metabolism. Before a test, therefore, food is withheld for at least 12 hours, the body protected from cold, and a period of rest and relaxation maintained for at least 20 minutes to one half-hour. It has been shown that heat production is 8 per cent. less when a patient lies quiet than when sitting up quiet. Ingestion of normal amounts of food adds seven calories per hour for 24 hours in heat production.

Some of the endocrine glands play a very important part in the rôle of metabolism. Chief of these is the thyroid, which is the most important physiological regulator of the

* Read before the Congress of Anæsthetists, Atlantic City, May 25, 1925.

metabolic rate. When completely absent, basal metabolism approaches 40 per cent. below normal. The active principle of the gland is thyroxin, 2 mgms. of which administered daily will keep the metabolic rate 20 per cent. to 30 per cent. above normal; 3 mgms. daily, 50 per cent. above normal.

The effect of the adrenal glands through their active principle, epinephrin, is secondary to that of the thyroid. Its action is rapid and transient, and not prolonged as is the case with thyroid. The adrenal secretion is called forth by mental anxiety, and therefore a factor before operations.

The metabolic rate is found to be reduced in:—

Thyroid disease as myxœdema and hypothyroidism; cretinism, coilloidgoitre, and sometimes in malignant thyroid; malnutrition, hypopituitarism, diabetes, epilepsy, Addison's disease, arthritis; some skin diseases, as eczema; oophorectomy, sleep.

The metabolic rate is increased in:—

Thyroiditis, exophthalmic goitre, and toxic adenomata.

Severe anæmias of primary and secondary types, highest in pernicious. (Blood transfusion tends to lower the basal metabolic rate.)

Lukæmia.

Cardiac and nephritic diseases, especially if compensation is failing.

Fever—for each degree of which the metabolism is increased 7.2 per cent., irrespective of what disease is causing the fever.

During exercise the greatest normal increase is produced.

Effects of various drugs are as follows:—

Caffeine, 8 to 10 grs., raises the rate 7 to 23 per cent.

Atropine and camphor increase the rate.

NACL intravenously increases the O_2 absorption, with little effect on the CO_2 given off.

Pilocarpin gives 10 per cent. increase.

Phosporus—small doses elevate, toxic doses depress.

Aspirin—1 gr. increases the heat production 6.1 per cent. in normal cases. In febrile shows little effect.

Quinine usually retards.

Chloral Phenacetine Acetinilide—depress the metabolic rate.

Heroin Strychnine and Bromides of Barium and Radium —no effect.

In a dog experiment performed by one of us with ether, the oxygen consumption in light anæsthesia was 1500 c.c. in five minutes, and as the narcosis was deepened the oxygen consumption fell progressively until only 125 was absorbed in five minutes, when the dog was profoundly anæsthetized, again increasing when the narcosis was lightened. The same condition of reduction of the metabolic rate is obtained in clinical anæsthesia when ether is administered. Not enough of such estimates have been made, however, to determine the average reduction per square metre of body surface in the human, but from our tests and observations the reduction is much greater than under nitrous-oxide oxygen. The difference between these two anæsthetics may be due to the greater general reduction of oxidation throughout the body under ether, which seems logical from clinical evidence. It may be that nitrous-oxide affects the more highly developed nervous tissue.

Taschiro's research on the metabolism of excised living tissue throws some light on the subject, which is of interest to the anæsthetist. He showed that nervous tissue uses much more oxygen per unit mass than any other tissue in the body, and that the rate of CO_2 produced form the oxygen absorbed varied with the irritability of the tissue. In other words, that the rate of CO_2 production was a measure of irritability. From his work we are led to suspect that the brain uses more oxygen than any other tissue, and is more profoundly affected by the anæsthetic, or by any reduction in oxygen by which the anæsthetic accomplishes its purposes.

We might logically go farther, and say that if the brain was not more easily depressed than the heart, for example, that every patient would die at the moment the brain was sufficiently depressed in its metabolism to produce anæsthesia. That the difference in metabolism between the brain and other tissues represents the margin of safe anæsthesia. If this difference is small, as might occur in certain conditions, as fever, growth, cholæmia, or other toxæmias, the safe margin of anæsthesia is correspondingly narrowed. In a degree this might explain the narrow margin of some patients

which we see clinically. Whatever the difference of metabolism of various may be, it is quite certain that those anæsthetic agents investigated reduce the general metabolism of the patient roughly in proportion to the depth of anæsthesia and the kind of anæsthesia used. Taschiro showed that some local anæsthetics when applied to the nerve reduced its metabolism, which also seems to indicate that metabolic reduction is a function, if not the function of anæsthetic agents.

The administration of morphine before an anæsthetic may produce its good effect by acting upon the adrenals to prevent excessive secretion (which would further increase the metabolic rate), or by benumbing the sensibilities of the patient to his ordeal, and so lessen the discharge of epinephrine, which results from nervous excitement. But it is definitely known that the hyperthyroid does well or badly, as his metabolism is reduced or increased. It would appear to be our duty as anæsthetists to attempt to regulate the metabolic activity of these and other cases within certain limits, by the use of appropriate drugs and anæsthetics. When metabolism is too high, or tends to increase, to reduce it; and if too low, to increase it.

During an operation, and without making a metabolic test, which is difficult and impracticable as a general practice, how may a change in metabolism be detected? The answer —by the respiration in major part and by the pulse and other signs in minor. The breathing of the patient is in response to the use of oxygen, and the production of CO_2. If the respirations become rapid, metabolism is increasing, and if they become excessively slow, it is decreasing. Deeper or lighter narcosis often corrects it, but many times this remedy is not effective, and others should be employed.

Morphine in some combination may be more effective to reduce metabolism than the anæsthetic, or atropine may be needed to increase it. Certainly both morphine and atropine, one the antagonist of the other, are not indicated in the case at the same time. Our observations of morphine and atropine point to the conclusion that when given together the patient is not protected as intended from the fluctuations upward in metabolism during operations. Morphine and

hyoscine operate better together in this respect, but the dose must be properly chosen to perform its function.

In an operation for splenectomy performed April 11th, 1925, nitrous-oxide oxygen was administered for about half the time, and ethylene-oxygen for the remainder, given through the united gas oxygen apparatus and metabolor. In repeated tests the rate of absorption under gas oxygen was greater than under ethylene oxygen, although the average difference amounted to only about 1600 c.c. per hour. In this same case on the morning of the operation his basal metabolism was 19 per cent. above normal—a usual condition for splenic anæmia. Taken again one hour after the administration of H.M.C. No. 1 (morphine $\frac{1}{4}$ gr. hyoscine 1/100 gr.), the metabolic rate was 7 per cent. below normal—a difference of 26 per cent., apparently due to the hypnotic. The average reduction of metabolism is less than 26 per cent. after a H.M.C. No. 1, more commonly about 10 per cent.

In another case, a hyperthyroid with a metabolic rate of 51 per cent., a H.M.C. No. 2 (morphine 1/6 gr. hyoscine 1/200 gr.) was given. One hour later the rate was still 51 per cent. Under local anæsthesia, the left thyroid artery was ligated at this time. A few minutes later the metabolic rate was 73 per cent. This shows she was not sufficiently protected. In fact, the H.M.C. No. 2 had accomplished nothing, either as to effect on metabolism, or the usual clinical effects, noted from an adequate dose of this drug. Nitrous oxid-oxygen with such a light dose of morphine and hyoscine usually controls better. An adequate dose of morphine, however, is better whether the anæsthetic is local or general.

In this brief summary of clinical investigation, and the work of others, we would emphasize the following points :—

1. That metabolism is depressed with certain anæsthetics commonly used.
2. That this reduction of metabolism within certain limits is protective to the patient and may be *the* desirable factor in anæsthesia.
3. That the respiration of the patient is a good clinical guide regarding the changes in metabolism during narcosis and operation.

4. That drugs should be used to assist the anæsthetic in controlling metabolism—morphine to decrease it, and atropine or others to increase it, as required during the operation.
5. That the dose of drugs employed should be large enough to produce the desired effect.

BOOK REVIEWS.

The third edition of Arthur E. Hertzler's "The Technique of Local Anæsthesia" (Henry Kimpton, London, 25/- net) brings a very authoritative work up to date. While retaining all the leading features of the original volume, the latest edition presents the essential features in small space, which makes it easier for the busy reader to get to the kernal of the subject treated. One is pleased to note that Hertzler wastes little time on non-essentials; one quite agrees with him in his assertion that local anæsthesia should no longer be regarded as a stunt to be performed as an athletic event.

The author quite rightly devotes most space to his own methods. This avoids confusion and does not hamper the beginner. Writers frequently add only padding to a work by describing again methods which have appeared elsewhere, and which he generally damns with the faintest praise. In a subject of this character descriptions should be short, sharp and crisp, and this book can be commended for following this plan.

The illustrations are plain and good. They show only what the reader is anxious to see. Hertzler proves that he understands what is really required in this direction when he states, in his preface, somewhat picturesquely, that he disapproves of "the striving after melodramatic effect by presenting pictures of patients wearing a smile while their guts are being slashed about."

This is a good work to have in one's library, as it covers the entire field of local anæsthesia authoritatively and briefly, and contains the minimum of padding.

"Medical Education" (the Macmillan Company, New York, $2.50, for the General Education Board) is a survey of medical education during the last fifteen years. Abraham Flexner, the author, who is a brother of the gifted Simon Flexner, has succeeded in covering every portion of Europe and America in this survey, and, from a world of first-hand information, he draws inferences which should prove of great value to those responsible for the teaching of medicine on both sides of the Atlantic.

At the request of the Editor, the following abstracts of papers read at the Congress of Anæsthetists, Atlantic City, May 25th, 1925, have been prepared by the authors themselves.

THE VALUE OF HELIUM-OXYGEN ATMOSPHERE IN DIVING AND CAISSON OPERATIONS.

By R. R. Sayers,

Chief Surgeon, Department of Interior, Bureau of Mines; Surgeon, U.S. Public Health Service,

and W. P. Yant,

Associate Chemist, Department of Interior, Bureau of Mines, Pittsburgh Station.

The Bureau of Mines is interested in the problem of the health hazards encountered by men who work in compressed air in tunnel construction and other engineering operations. This is also of interest to the Navy Department on account of the possibilities for extending the range of salvaging and marine-engineering operations. The most important hazards are oxygen poisoning, produced by breathing high partial pressures of oxygen, and caisson disease.

By controlling the amount of oxygen present at any pressure, ill-effects can be avoided. As an example, if the oxygen present is reduced to 5 per cent. at 1 atmosphere a pressure of slightly more than 4 atmospheres is needed to bring the equivalent partial pressure of oxygen to normal, 20.9 per cent., and the concentration produced by 10 or 12 atmospheres would cause no harmful effects due to oxygen.

Caisson illness is a condition resulting from too rapid decompression of an individual after having been exposed to atmosphere at high barometric pressure. Bubbles of nitrogen are found in the body tissue and fluids during decompression or ascent in diving. Any gas that has a lower coefficient of solubility or (and) a greater diffusivity than nitrogen would tend to reduce the danger of caisson disease, as well as reduce the period of decompression for a given condition of exposure, but such a gas in addition would have to be inert like nitrogen as regards physiological effects. Dr. J. H. Hildebrand called the authors' attention to the fact that the coefficient of solubility of helium had recently been determined to be less than that of nitrogen, and that it might possibly be of use in caisson illness. In addition to the advantage of lower solubility, it appeared to the authors that the greater diffusivity of helium would be an additional advantage, resulting in more rapid and easier escape from the tissues and body fluids during decompression.

Accordingly the authors, who had been considering the compressed air problems, included tests in helium on their studies. Comparative experiments were conducted on white rats and later on guinea pigs with synthetic atmospheres of nitrogen and oxygen and of helium and oxygen, the results of which give data as to their relative value.

From a comparative examination of all data as regards to decompression periods and symptomatic and pathological findings, it is

evident that, with similar exposure and decompression periods, the condition of the animals exposed to the helium-oxygen mixture is much better than that of those exposed to the nitrogen-oxygen mixture. It may be safely said that for similar effects the advantage of the lessened decompression time of helium over nitrogen is somewhere in the order of 1 to 3 or 4.

In order to ascertain whether any discomfort would be caused from breathing helium, the gas was inhaled by several of the investigators for periods up to two hours. There was no noticeable effect, except a temporary rise in the pitch of the voice. The gas was found to be as agreeable and pleasant as normal air.

The possibilities of the use of the helium-oxygen atmosphere may be for extended depth and time of submarine operations; also for the prevention of caisson disease in tunnel and caisson workers, and by using the mixture as a wash gas during decompression among tunnel workers.

THE ADRENALS AS DOMINANT FACTORS IN TISSUE RESPIRATION AND THEIR BEARING AS SUCH UPON ANÆSTHESIA.

By Charles E. de M. Sajous,
Professor of Endocrinology, University of Pennsylvania.

Recalling that the leading fundamental problem of medical science, the nature of tissue oxidation (including its corollaries fever, hæmolysis, autolysis, etc.), is still unsolved, as indicated by Prof. Halliburton's statement (1921) among many others, that "our knowledge of tissue respiration is so scanty that we can say but little of its pathological bearing," Dr. Sajous submits that this obscurity, which retards correspondingly the study of the effects, normal and pathological, of anæsthetics, may be eliminated by advanced knowledge of the functions of the ductless glands.

He points out that the independent researches of many physiologists, biochemists, pharmacologists and clinicians, considered collectively, have sustained his own interpretation of the functions of the ductless glands and that several of these organs are the dominant fundamental factors in tissue respiration and thermogenesis.

According to Dr. Sajous, the adrenals carry on these functions by means of their three dominant constituents, one which he termed "adrenoxidase" in 1907, and the familiar lipoids, lecithin and cholesterol. (1) *Adrenoxidase,* the secretion of the adrenal medulla (the active principle of which is adrenalin) is a catalytic enzyme forming part of the hæmoglobin which enables this pigment to become converted into oxyhæmoglobin on exposure to the pulmonary air, and to supply the tissues with activated oxygen; (2) *Lecithin,* a product of the adrenal cortex is a lipoid containing 3.88 per cent. of phosphorus, found in all tissues (in the brain as cephalin, in the heart as cuorin, etc.) which is oxidized catalytically by the adrenoxidase

and thus liberates heat, acting as " thermogen "; and (3) *Cholesterol*, a monatomic alcohol, also found in all tissues, which controls as " moderator " the thermogenic activity of lecithin. It thus acts as the " physiological antipyretic " (also as antihæmolytic, as is well known) and as " antiautolytic " by preventing excessive heat production, when fever, produced by the adrenoxidase-lecithin reaction exceeds safe limits.

The *erythrocytes*, which are well known to contain adrenoxidase, lecithin and cholesterol, are replenished periodically with these substances by passing through the adrenals, in order to deliver them to the tissues.

Bearing also upon anæsthesia is the lymphatic system, and notably, insofar as fatalities are concerned, status lymphaticus and thymic death. Dr. Sajous points out that the lymphatic circulation is also dominated by the adrenals. Recalling that adrenalin produces contraction of unstriated muscular fibres of the heart and arterioles, he states that such fibres are also known to exist in the capsules and trabeculæ of all lymph glands and nodes, and that they receive arterial blood as do other tissues and therefore its adrenal products. Operating as do the lymph hearts of many lower forms including mammals, the lymph glands by periodical contractions, about one per minute, propel the lymph, perpetuating its circulation. Deficient adrenal activity by slowing the lymphatic circulation leads to engorgement of all lymphatic organs, including the thymus. This condition is the starting point of status lymphaticus and may lead to thymic death through pressure upon the trachea.

Among the elucidative features of these principles in the practice of anæsthesia, Dr. Sajous urges the importance of the non-striated fibres in both the vascular and lymphatic systems. Thus, in cases with deep circulatory depression, shock, etc., the large intravenous injections of saline solution used act not only on the blood circulation, but also on that of the lymph, preventing engorgement in the lymph vessels and glands, and also in the intracellular spaces, thus sustaining the life process itself. If, simultaneously, adrenalin, or better, pituitrin, which contains the active agent of both the cortex and medulla, be injected, the tone of the heart and of both the blood and lymph vessels is promptly restored owing to their stimulating action on the non-striated muscular fibres in their contractile mechanisms. Tachycardia is also antagonized by this treatment, being due, in many cases, to the existing general vasodilation. Both digitalis and strychnine owe their tonic effects on the heart in part to their stimulating action on the adrenals; hence the beneficial effects of these agents. The need and effects of warmth of the body to sustain the vital process during anæsthesia, to co-operate with the thermogenic action of the adrenal secretory products, is also emphasized. These various indications apply only to asthenic cases in which circulatory depression prevails. The action of various anæsthetics on the blood

lipoids, and also their relative behaviour in the presence of various diseases of the heart, blood vessels, lymphatic system, liver and kidneys, including danger signals, are also studied in the light of the principles submitted, along with what newer measures of treatment they suggest.

STUDIES OF THE PHYSIOLOGICAL ACTION OF SOME UNSATURATED HYDROCARBONS.

By LLOYD K. RIGGS, Ph.D., and HAROLD D. GOULDEN, B.Sc.
(From the Research Laboratories of E. R. Squibb & Sons, New Brunswick, New Jersey.)

The use of ethylene and acetylene gases as anæsthetics has made it appear desirable that a general study of the physiological properties of unsaturated hydrocarbon gases be undertaken.

The inhalation of hydrocarbons in the following concentrations induces anæsthesia in white rats (100 to 150 grams) in fifteen minutes; ethylene 90 per cent., propylene 40 per cent., butylene 20 per cent., amylene 6 per cent., and acetylene 78 per cent. These concentrations of the various hydrocarbons are hence considered to be of equal anæsthetic potency. If the anæsthetic potency of propylene is taken as unity, the potencies of the other hydrocarbons then become: ethylene 0.44, propylene 1.0, butylene 2.0, amylene 6.6, and acetylene 0.52.

Butylene and amylene differ from ethylene, propylene and acetylene in that they produce marked nervous symptoms during the induction stage of anæsthesia.

The inhalation of methylacetylene 5.0 per cent. or of propadiene 20 per cent., for ten to fifteen minutes, causes prostration of white rats, but such violent nervous symptoms are produced by these hydrocarbons, that they can scarcely be said to possess anæsthetic properties.

The inhalation of hydrocarbons in the following concentrations causes death of white rats (100—150 grams) in about two hours: propylene 65 per cent., butylene 20 per cent., amylene 6 per cent., acetylene 90 per cent., methylacetylene 5 per cent., and propadiene 15 per cent. On the basis of the toxicity of propylene as unity, the toxicities of these hydrocarbons are, butylene 3.25, amylene 10.8, acetylene 0.73, methylacetylene 13, and propadiene 4.3. The toxicity of ethylene cannot be determined at atmospheric pressure on account of its low potency, thus 90 per cent. ethylene is required to induce anæsthesia in white rats and the toxicity of higher concentrations is complicated by oxygen starvation.

The anæsthetic potency and toxicity of the hydrocarbons within a homologous series are shown, above, to be dissimilar functions of the molecular weight. The potency and toxicity of each hydrocarbon may be related in the form of a ratio, thus: $\frac{\text{Potency} \times 100}{\text{Toxicity}} = \text{Index}$.

The index thus obtained may be called the index of safety or anæsthetic index. The indices for the several hydrocarbons are as follows: Propylene $\frac{1 \times 100}{1} = 100$, butylene $\frac{2.0 \times 100}{3.25} = 61.0$, amylene $\frac{6.6 \times 100}{18.8} = 61.0$ and acetylene $\frac{0.52 \times 100}{0.73} = 71.0$

Animals (rats) deeply anæsthetized for 7½ hours with 55 per cent. propylene, recovered within two minutes of their removal from the gas mixture and showed no bad after-effects. Pregnant female rats anæsthetized (propylene) daily during the first two weeks of gestation bore normal young. Fifteen human subjects have been anæsthetized by various concentrations of propylene (30 per cent. to 85 per cent.) and report that their experiences were not unpleasant. None of the subjects felt nausea.

The relatively high potency of propylene, which admits of its use with adequate supplies of oxygen, its freedom from immediate or delayed toxic action in moderate concentrations, and the wide range between the minimum anæsthetic and minimum toxic concentrations of propylene, appear to favour the consideration of this gas as a clinical anæsthetic.

THE CHEMISTRY AND PHYSICS OF DRY NITROUS OXIDE.

By Walter S. Crowell, B.Sc.,

Chief Chemist, the S.S. White Dental Mrg. Co., Philadelphia.

After briefly describing the generation and purification of nitrous oxide, the dehydration process is described in detail.

Drying must be carried to a point such that no water will be deposited from the gas at normal temperatures or at the lower temperatures resulting from the expansion of the compressed gas and the boiling of the liquid gas during administration. If water is deposited in the administering apparatus at the point of pressure reduction a frost-like solid will form, consisting chiefly of $N_2O.6H_2O$, which will obstruct the flow of the gas, and make necessary constant adjustment of the apparatus by the operator.

To remove water, the gas is pumped through suitable traps, then through a dehydrating train containing calcium chloride and other more powerful dehydrating agents such as lime, fused caustic or phosphorus pentoide. After passing through the dehydrator the gas is compressed into the cylinder. The cylinder is attached to the pump through the dehydrating apparatus, thus the cylinder pressure and the pressure in the dehydrator are the same. The dehydrators are kept above the critical temperature of the gas to insure against too early liquefaction.

When filling begins little water is removed by the traps, the chemical dehydrating agents doing most of the work. As the pressure increases more and more water is trapped out and less required to be removed by the dehydrating agents.

It is necessary to dry the cylinders before filling with dried gas.

PROCEEDINGS OF SOCIETIES.

At the May 9th, 1925, meeting of the Scottish Society of Anæsthetists, held at Dundee, Dr. A. Mills read his Presidential Address on the subject of

The present position of chloroform.

Doubtless we have been slow in this district to accept the view that the administration of anæsthetics is work requiring special skill and experience, for it is only within very recent times that the staff of anæsthetists in our hospital has been adequately increased. There are now six anæsthetists, a number sufficient to overtake practically all the work that is being done. I fear, however, that a long time may yet elapse before there will be a like demand for the services of the anæsthetist in private practice. Strange as it may seem to us, many a doctor still holds the opinion that "anyone can give chloroform." The economic factor, too, is I fear strong enough to perpetuate the old customs, and we may accept the fact, that for some time to come, many private patients will be anæsthetised by comparatively inexperienced administrators. It is with this fact constantly in mind that I have framed the views and opinions which I am about to express. As teachers our duty is to impart that knowledge and instruction to our students which will make them safe and efficient administrators of anæsthetics, in so far as safety and efficiency is possible with the meagre opportunities afforded by ordinary general practice.

Frequently at our meetings in the past the value of chloroform as an anæsthetic has been discussed. In 1919 Dr. Paton Boyd took this subject for his presidential address, and severely criticised the extreme attitude of the American anæsthetists. The Committee of Anæsthesia of the American Medical Association reported in 1912 that the use of chloroform was no longer justifiable, and that its use for minor operations should cease, but that it was sometimes of convenience for inducing anæsthesia in alcoholics or in difficult cases. I am not aware whether a further report on this subject has been issued from this source. In some places it would appear that chloroform has been entirely discarded. During 1923, 17,813 anæsthesias were administered at the Mayo Clinic and chloroform was not used on a single occasion.

Are we prepared to clearly define our own views on this important subject? When that report was issued by the American Medical Association in 1912, chloroform was practically the only anæsthetic used in Dundee and the surrounding district for all ordinary operations; indeed, we were not asked to give an anæsthetic, we were asked to "give chloroform." Though many practitioners now realise that there are certain limitations to the use of chloroform and that other and safer anæsthetics are available, others persist in the belief that

provided with nothing but chloroform, they are justified in proceeding to induce and maintain anæsthesia wherever and whenever general anæsthesia is required. Chloroform deaths thus still take place and by " chloroform death " I mean a very definite form of fatality. I suggest that our knowledge of the pharmacological action of chloroform and other anæsthetic drugs is now such that these deaths are avoidable, and that we as teachers of anæsthetics are responsible to inculcate better and safer methods. Such methods, however, must be simple ones and not associated with the use of complicated apparatus, if they are to be adopted by the practitioner desirous of giving an anæsthetic only when the necessity arises in his own practice. Some such simple method I shall attempt to describe, but before doing so I should like to discuss briefly the manifest contra-indications to the use of chloroform.

Chloroform of course possesses certain obvious advantages. It is a powerful anæsthetic and easy to administer. A few cubic centimetres of chloroform administered on a towel or piece of lint are sufficient to produce anæsthesia. It is non-irritating to the respiratory passages and is pleasant to inhale. It produces a quiet type of anæsthesia and no undue congestion of the incised parts. The dangerous effects of chloroform fall into four groups—(1) depression of the respiratory centre, (2) fall in the blood pressure, (3) late toxic effects—delayed chloroform poisoning, (4) chloroform syncope—death occurring very suddenly, usually but not invariably in the stage of induction.

I have not mentioned these groups in the order of their importance, the most important one being Group 4, which I should prefer to discuss last.

(1) Chloroform has a depressant effect upon the respiratory centre. It dulls this centre's sensitivity to its normal stimulus from carbon dioxide. During anæsthesia the amount of carbon dioxide produced in the body is considerably reduced and if at the same time the centre become less sensitive a condition of anoxæmia may result from deficient ventilation of the lungs. This danger is further increased by the fact that chloroform also reduces the oxygen-carrying capacity of the red corpuscles. Depression of the respiratory centre is usually of gradual onset, and manifests itself in an unmistakable manner. The careful and observant administrator rarely permits the condition to become serious. Moreover, depression of the respiratory centre is not an effect peculiar to chloroform. The other anæsthetic agents have a similar action, but in less degree. It cannot thus be regarded as a factor of sufficient importance as to entirely contraindicate the use of chloroform.

(2) Chloroform in full anæsthetic doses causes a fall in blood pressure, but this fall is roughly proportionate to the depth of anæsthesia reached. The low blood pressure resulting from chloroform anæsthesia is similar in nature to that of surgical shock. It is

not due to failure of peripheral resistance for the arterioles are in a state of constriction. It is due to an imperfect filling of the heart in diastole and consequent diminished output into the arterial circulation with a stagnation of blood in the splanchnic area and in the capillaries. A tame rabbit suspended by the ears passes into a state of shock and dies. This condition of gravity shock is much more easily produced in the chloroformed animal than in the animal which has not been chloroformed, and is the more easily produced the more deeply the animal is anæsthetised.

It has been shown that quantities of histamine too small to produce any effect upon a normal animal cause a marked fall of blood pressure when injected into an animal after prolonged chloroform anæsthesia. The histamine-like bodies produced when tissues are injured are believed to be the chief cause of shock, so that this action of chloroform in increasing the susceptibility of the tissues to histamine will also account for its predisposing to shock.

Chloroform is thus strongly contraindicated in cases where the patient is already suffering from a low blood pressure. In this respect patients suffering from shock, or who are in a gravely debilitated condition, are unsuitable subjects for chloroform anæsthesia.

(3) Post-anæsthetic toxæmia may follow the administration of any anæsthetic but is much more frequent after chloroform. The condition is also known as delayed chloroform poisoning, and its symptoms are familiar to all of us. It is much more common in children than in adults and toxæmia and starvation are powerful predisposing factors. It is a condition of acid intoxication comparable to that met with in diabetes, salicylate poisoning, and recurrent or cyclic vomiting. Acidosis or acid intoxication is due to the presence in the circulation of an abnormal amount of substances of the acetone group, and this excess may be due to too rapid formation or to too slow elimination or to both. Acetone bodies are the result of increased fat metabolism, which again is a result of the diminution in the carbohydrate metabolism. Elimination is performed through the liver and kidneys, and deficient elimination results from damage to liver and kidney cells. Starvation and constipation and an anæsthesia increase fat metabolism and increase acetanæmia. Septic absorption and chloroform damage liver and kidney cells. Clinically we find that the child who is suffering from a septic abdomen and who has had little or no food for a few days prior to operation, is the most susceptible of all patients to post-anæsthetic acidosis. Chloroform is absolutely contraindicated in these cases. It is also contraindicated in the case of children known to suffer from cyclic vomiting. Such children may have a severe and fatal attack precipitated by a very short administration of chloroform. There is no evidence that chloroform produces an increased acetonæmia more readily than does ether. In this respect anæsthesia and asphyxial conditions are closely related. There is however, strong evidence that chloroform, by its

toxic effect on the liver and kidney cells, is much more dangerous than ether, and the acetonæmia produced is maintained and aggravated through deficient elimination. Beesley, in 1906, referring to work in the Children's Hospital in Edinburgh, reported that " out of nineteen cases of acute appendicitis operated on under chloroform fourteen died, out of twenty-four cases operated on under ether only two died, and that not as the result of acid intoxication."

(4) The large majority of chloroform deaths (80 per cent. or more) take place during the induction period. The victim is not infrequently a robust adult male. As an example of what may be regarded as a typical chloroform death, I quote the following case :—" The patient was an ex-soldier of 53 years of age who had had thirty years' service abroad. He was of spare muscular frame, arteries hard, chest somewhat emphysematous and rigid, heart sounds could only be faintly heard, but his general condition was good. The operation intended was stretching the sciatic nerve. Anæsthesia was induced with chloroform on an open mask. The stage of excitement was very prolonged with violent struggling spasm of the muscles, irregular respiration and some cyanosis. Suddenly there was general muscular relaxation and the breathing lost its spasmodic character.. The anæsthetic was at once withdrawn. Then followed a few deep regular respirations, but the breathing soon became shallow and stopped, the pupils being widely dilated and the complexion of a livid hue. No pulse could be felt at the wrist and the heart sounds were inaudible. Attempts to resuscitate the patient failed." It is obvious that the patient's heart had suddenly ceased to beat and respiration only stopped after the respiratory centre had failed from want of blood. I believe that I am correct in stating that a similar sequence of events never takes place when ether is the anæsthetic employed. Of a series of 33 chloroform deaths which I have investigated, I find that at least 21 were of this nature. Such deaths are due entirely to the misuse of chloroform, and should cease to take place if our teaching is sound and is honestly followed. Of the several rival theories advanced to account for these sudden chloroform deaths, two only appear to me to be of value, and to be more or less supported by clinical experience. Violent excitement in the induction stage will be admitted by all to be a frequent precursor to a catastrophe. In the series of cases investigated, this state of excitement was emphasized in the report in 12 out of the 21, and may of course have occurred in others. Pehl found in a dog, which had been killed by a few breaths of concentrated chloroform vapour, that the blood in the left ventricle contained .2 per cent. chloroform, whilst that in the right ventricle contained only .02 per cent. He suggested therefore that in many cases death was caused as follows : During the stage of excitement and irregular spasmodic respiration, the patient first held his breath and allowed chloroform vapour to accumulate under the mask : then he took a deep breath inhaling a concentrated chloroform vapour which passed

into the blood and went direct to the heart and coronary arteries. In this way a toxic action upon the heart was produced long before the other tissues of the body were saturated with the drug. This theory would appear to fit in with a number of reported cases and could reasonably be applied to 12 or more of my series of 21. Levy, on the other hand, maintains that this theory has no experimental support, and states that high percentages of chloroform administered to animals are incapable of producing sudden stoppage of the heart. The matter would appear to require further investigation.

(To be continued.)

ABSTRACTS OF CURRENT ANÆSTHETIC LITERATURE.

Spinal anæsthesia with stovaine and alypine. G. E. VAQUIE. *Presse Medicale,* 28th March, 1925.

Some 16 years ago, encouraged by the records published by Jonnesco, I commenced my series of 2,500 cases and I have now evolved a very simple formula which has given me excellent results without any fatal accidents attributable to the method.

In my studies I have been struck by the diversity of opinion, some surgeons favouring this form of anæsthesia while others regard it as dangerous; my early cases were anæsthetised by a mixture of stovaine and cocaine, but owing to the frequent occurrence of alarming symptoms I abandoned cocaine for alypine and have adopted the following formula:—Stovaine, O.G. 75, Alypine, O.G. 15, Strych. Sulph., O.G. 001, to make 10 ampoules.

For sterilising, alypine should be submitted four times to a temperature of between 60 and 70 degrees, but stovaine may be brought to a temperature of 100 degrees. Alypine is soluble in water, and in a 2 or 4 per cent. solution it keeps well but not in weaker strengths; its toxicity is low, but given intravenously it is dangerous; it is a vaso-dilator, and patients retain a good colour and are saved from post-operative faintness. As to stovaine we regard it as a cardiac stimulant, and on that account superior to other analgesics.

The three sites for injection which I have used are 1-2 D.V., 3-4 L.V., and 4-5 L.V. The first position, for anæsthetising the thorax with half the usual quantity of the drug, I have found unnecessary as the same end can be achieved by lowering the head end of the table for a minute after the ordinary lumber injection.

Incidents such as vomiting and nausea are quite the exception after stovaine and alypine; with some patients there is pallor, sweating, or slight paralysis of the anal sphincter, but these phenomena are transient and trivial.

The following unique occurrence took place in a man who had been injected in the 2-3 D. space. After repeated yawning the respiration ceased, the eyelids remained closed but the corneal and pupil

reflexes persisted. When told to imitate our breathing he did so, but again ceased to breathe until the order was repeated, after which he recovered.

For paralysis of the bladder I employ sounds for one or two days and administer strychnine.

It is usual to find the temperature 1 or 1½ degrees above normal on the day following operation.

I regard as very important the post-operative treatment, and keep my patients in a darkened ward in the horizontal posture allowing no movement or conversation, and not permitting any fluid to be drunk during the first three or four hours.

Three cases of syncope during anæsthesia treated by intra-cardiac injection of adrenalin; one permanent, two temporary recoveries. TASSO ASTERIADES. *Presse Medicale*, 13th May, 1925.

According to physiological and clinical observations an interval of time occurs between apparent and actual cardiac death, which being about ten minutes, permits of various remedial measures being employed; after fifteen minutes success is improbable, after twenty-five minutes failure is certain as by that time the vitality of the nervous centres has become irremediably damaged; in actual practice the available time is probably not longer than five minutes, for which reason it is important to act quickly and to avoid wasting time in such measures as artificial respiration, which should be regarded as a complement to the only reliable method of ccardiac resuscitation, namely, intra-cardiac injection of adrenalin.

Puncture of the heart muscle is in itself free from danger. Toufet and Baumann could find no trace of the puncture macroscopically in post-mortems which they made, although in one of their cases the heart had been punctured six times in two hours; there was not even any trace of blood in the pericardium. Patients who recovered showed nothing abnormal except, in some instances, an insignificant degree of tachycardia.

It is generally agreed that for the injection adrenalin is unrivalled. We have reports of 34 cases of which 18 survived, an average of 52.9 per cent.

The three cases reported here were as follows :—

(1) Man, aged 22, radical cure of hernia under chloroform; syncope at commencement of operation. Artificial respiration with injection of heart two minutes after collapse. Recovery.

(2) Boy, aged 5, sarcoma of lower jaw. Under chloroform collapse at end of operation. Artificial respiration; injection of heart eight minutes after syncope; return of pulsation for few moments; second injection, again temporary pulse. Death.

(3) Woman, aged 28. T.B. peritonitis under chloroform; syncope after deepened anæsthesia for suturing; heart massage and injection 10 minutes after collapse; revival for five minutes. Death.

A. L. FLEMMING.